

Deliberate overfeeding in women and men: energy cost and composition of the weight gain*

BY GILBERT B. FORBES¹, MARILYN R. BROWN²,
STEPHEN L. WELLE³ AND BARBARA A. LIPINSKI⁴

Departments of ¹ Radiation Biology and Biophysics, ² Pediatrics, ³ Internal Medicine, and ⁴ Dietetics, University of Rochester School of Medicine and Dentistry, Rochester, New York 14642, USA

(Received 23 August 1985 - Accepted 13 January 1986)

1. Thirteen adult females and two males were overfed a total of 79-159 MJ (19000-38000 kcal) during a 3-week period at the Clinical Research Center, Rochester. The average energy cost of the weight gain was 28 kJ (6.7 kcal)/g, and about half the gain consisted of lean body mass (LBM) as estimated by ⁴⁰K counting.

2. A survey of the literature disclosed twenty-eight normal males and five females who had been overfed a total of 104-362 MJ (25000-87000 kcal) under controlled conditions: twenty-five of these had assays of body composition, and three had complete nitrogen balances.

3. When these values were combined with those from our subjects (total forty-eight), there was a significant correlation between weight gain and total excess energy consumed (r 0.77, P < 0.01) and between LBM gain and excess energy (r 0.49, P < 0.01). Based on means the energy cost was 33.7 kJ (8.05 kcal)/g gain and 43.6% of the gain was LBM; from regression analysis these values were 33.7 kJ (8.05 kcal)/g gain and 38.4% of gain as LBM.

4. Individual variations in the response could not be explained on the basis of sex, initial body-weight or fat content, duration of overfeeding, type of food eaten, amount of daily food consumption or, in a subset of subjects, on smoking behaviour.

5. The average energy cost of the weight gain was close to the theoretical value of 33.8 kJ (8.08 kcal)/g derived from the composition of the tissue gained.

The relation of energy intake to body-weight status is a topic of current interest. When human subjects are studied under controlled conditions energy deficits lead to weight loss, and the greater the deficit the greater is the loss. Does the converse hold true for excess energy intake, and do such gains involve fat or lean, or both? Do women gain weight more easily than men? Do thin people gain weight less easily than those who are overweight?

A number of short-term overfeeding studies have been done on normal human subjects during the past 50 years, ranging from the early studies of Cuthbertson *et al.* (1937) to the most recent one of Dallosso & James (1984*a, b*), and these have shown that nitrogen retention occurs as weight is gained. However, longer-term studies (of 2 weeks or more) provide for better quantification of the energy cost of weight gain and of the induced changes in body composition. Several studies have been reported in which subjects were fed under controlled conditions and observations were made on changes in body composition for periods of 2-7 weeks (Wiley & Newburgh, 1931; Passmore *et al.* 1955*a, b*; Miller & Mumford, 1967*a, b*; Goldman *et al.* 1975; Norgan & Durnin, 1980; Webb & Annis, 1983). Almost all the subjects were males.

The present report describes our studies of deliberate overfeeding of human subjects and uses these findings, together with those reported by others, to examine the questions posed previously. Overall, these studies encompass a wide range of energy intakes and of duration of overfeeding, a variety of diets, a range of initial body-weights, and include both sexes.

* Supported by grants HD 18454, RR00044, and AM32562 from the National Institute of Health, and based on work performed under contract no. DE-AC02-76EV03490 with the Department of Energy, at the Department of Radiation Biology and Biophysics, University of Rochester; assigned report no. UR-3490-2479.

SUBJECTS AND METHODS

Normal young adult subjects (two males, thirteen females) were housed in the Clinical Research Center, Rochester, for 24–28 d. They ranged in age from 18 to 41 years, in weight from 44 to 93 kg, and in body fat content from 6 to 25 kg. None had had a significant change in body-weight during the past 2 years. During the first week they were given a mixed diet in amounts designed to maintain body-weight; beginning with an intake equal to $1.5 \times$ basal metabolic rate (BMR), adjustments were made as necessary. The mean change in weight for the fifteen subjects during this week was -0.11 kg (range $+0.3$ to -0.6 kg), which is not significantly different from zero ($P = 0.19$). The average energy intake of the subjects during this week was considered to be their maintenance requirement. They were then given an additional 3 MJ (717 kcal)/d for 2 d, followed by an extra 5.0–7.5 MJ (1195–1793 kcal)/d for the next 15–19 d, making a total of 17–21 d of overfeeding.

The protocol was approved by our Institutional Committee on Human Investigation.

Diet

All food was prepared in the metabolic kitchen; all items were weighed, and energy content estimated from standard food tables. The maintenance diet provided 15% of energy from protein, 35–40% from fat and 45–50% from carbohydrate. The excess food provided 6% of energy from protein, 45–50% from fat, and 45–50% from carbohydrate. All food and drink were consumed in the Clinical Research Center, and any food not eaten at a given meal was added to the next meal. Sodium intake was held constant at 170 mmol/d. None of the subjects vomited or had diarrhoea. Faecal energy losses were assumed to be 5% of intake, which is the average value reported by Goldman *et al.* (1975) and Dallosso & James (1984*a, b*) for their overfed subjects.

Activity

The subjects were encouraged to be up and about, and to take walks in the hospital grounds or to exercise on a treadmill (4 km/h, zero grade) for 20 min twice daily. Some attended classes at the university (0.7 km from the hospital); others worked at clerical jobs part-time. Their activity patterns fell into the category of 'light physical activity' (World Health Organization, 1973). Every effort was made to keep each individual's activity pattern the same throughout the entire stay at the Clinical Research Center. Four of the female subjects were heavy smokers, and continued to smoke their usual quota of more than twenty cigarettes daily during their stay.

Procedures

Body-weight was measured each morning, after voiding and before breakfast, on a beam scale accurate to within 10 g. Body composition was estimated by ^{40}K counting (Forbes *et al.* 1968) on two occasions during the week of maintenance diet, and weekly during the overfeeding period. Lean body mass (LBM) was calculated on the basis that this body component contains 68.1 mmol K/kg in males and 64.2 mmol K/kg in females. BMR was measured in the morning after a 12 h fast, the subjects not having risen from their beds except to toilet, and having rested quietly for 30 min. Those who smoked cigarettes were instructed to refrain from smoking during the preceding 12 h. Measurements were made by the Noyons diaferometer (Kipp & Zonen, Delft, Holland), which operates on the principle that changes in oxygen and carbon dioxide concentrations produce changes in the electrical resistance of heated platinum wires. The flow-rate of air through the hood surrounding the subject's head was maintained at 50 litres/min. Ten readings were made, at 1 min intervals, and a correction was made for the respiratory quotient. Two assays were done during the first week, and once weekly thereafter. The results of a number of hormonal and metabolic studies will be the subject of a separate report.

LITERATURE SOURCES

Those reports were accepted which met the following criteria: (1) an initial period of weight maintenance before overfeeding; (2) the consumption of at least 100 excess MJ (24 excess Mcal) during the overfeeding period, thus excluding short-term studies; (3) a controlled environment, with food intake monitored; (4) an adequate protein intake; (5) assays of body composition before and at the end of the overfeeding period, or complete N balance; (6) normal health and nutritional status.

Only five of Miller & Mumford's (1967*a, b*) fifteen subjects met these criteria, and for these five the N balance values were incomplete; however, these authors did record an average increase in total body K, but details are not given. Two of Webb & Annis' (1983) eight subjects were excluded because of excessive weight changes during the weight-maintenance period. The findings of Olefsky *et al.* (1975) on overfeeding were not included because body-weight changes during the control period were not stated and because of unspecified variations in excess food intake.

A total of thirty-three subjects (five females, twenty-eight males) who met the previously-stated criteria were identified from the reports of several investigators (for sources, see Figs. 1 and 2, pp. 4 and 5). Twenty-five subjects had assays of body composition by densitometry before and at the end of overfeeding, and three additional subjects had determinations of N balance; for these three the change in LBM was calculated on the basis that this body component contains 33 g N/kg (Widdowson & Dickerson, 1964). These thirty-three subjects ranged in weight from 46 to 118 kg, and were overfed for periods from 14 to 83 d. Some were given excess carbohydrate, some excess fat and others a mixed diet. Total excess energy intake ranged from 104 to 362 MJ (24.8 to 86.5 Mcal).

RESULTS

Experimental subjects

Details are given in Table 1. All the subjects gained weight, the range being 3.5–5.8 kg, and fourteen of the fifteen had an increase in LBM. On average they gained 36 g/excess MJ (151 g/excess Mcal) consumed, and 51% of their gain consisted of LBM.

Weight gain occurred at a fairly uniform rate during the period of overfeeding. Although several subjects claimed that they usually gained weight before menstruation, the occurrence of menses during the study period had little effect on the course of weight gain. None took oral contraceptive drugs during the study period or for the week before admission to the Clinical Research Center.

There was no evidence of oedema, nor were there changes in serum electrolytes, serum protein or blood urea. Serum cholesterol levels rose slightly; the average increase was 0.51 mmol/l (19.7 mg/l). The average increase in BMR was 8.7 (SE 2.0)%, which was only marginally greater than the increase in body-weight of 6.0 (SE 0.4)%. There was a modest correlation between the increase in BMR and the increase in body-weight (r 0.63, $P < 0.05$).

Combined values: present results and literature values

Fig. 1 is a plot of weight gain *v.* total excess energy consumed during overfeeding for our subjects and those reported by others. Although there was considerable scatter in the values, the correlation coefficient was significant ($P < 0.01$), and the *y*-axis intercept was close to zero. This suggested that weight gain was in fact directly proportional to total excess energy intake, and that the linear function provided an adequate description of the relation. The average energy cost of the weight gain was the reciprocal of the regression slope, i.e. $1/0.0297 = 33.7$ kJ (8.05 kcal)/g.

Table 1. Details of individual subjects participating in the present experiment

| Subject no. | Sex | Wt (kg) | Height (m) | Initial body fat (kg) | Initial BMR (MJ/d) | Maintenance requirement (MJ/d) | Total excess energy (MJ) | ΔWt (kg) | ΔLBM (kg) | ΔBMR (MJ/d) |
|-------------|-----|---------|------------|-----------------------|--------------------|--------------------------------|--------------------------|----------|-----------|-------------|
| 1 | ♂ | 93 | 1.81 | 16 | 9.07 | 15.9 | 159 | 5.85 | 5.10 | 1.46 |
| 2 | ♂ | 70 | 1.80 | 6 | 6.16 | 12.1 | 110 | 4.30 | 3.00 | 0.39 |
| 3 | ♂* | 71 | 1.65 | 22 | 6.00 | 10.0 | 134 | 4.46 | 2.82 | -0.06 |
| 4 | ♀ | 50 | 1.64 | 9 | 5.40 | 9.91 | 123 | 5.21 | 2.38 | 1.18 |
| 5 | ♀* | 57 | 1.68 | 12 | 6.44 | 9.99 | 133 | 4.54 | 1.41 | 0.20 |
| 6 | ♀† | 58 | 1.72 | 12 | 5.59 | 8.78 | 123 | 3.48 | 2.12 | 0.29 |
| 7 | ♀ | 73 | 1.59 | 25 | 6.58 | 11.3 | 124 | 4.21 | 3.10 | 0.73 |
| 8 | ♀ | 55 | 1.65 | 15 | 5.53 | 9.36 | 128 | 5.20 | 1.40 | 0.40 |
| 9 | ♀ | 65 | 1.62 | 13 | 5.90 | 10.2 | 115 | 4.32 | 0 | -0.01 |
| 10 | ♀ | 75 | 1.67 | 17 | 5.98 | 9.33 | 145 | 4.45 | 0.51 | 0.72 |
| 11 | ♀ | 80 | 1.76 | 23 | 6.49 | 10.9 | 148 | 3.93 | 4.25 | -0.14 |
| 12 | ♀ | 63 | 1.75 | 11 | 6.04 | 9.45 | 135 | 4.14 | 3.22 | 0.31 |
| 13 | ♀* | 67 | 1.67 | 24 | 5.26 | 10.1 | 128 | 4.85 | 1.62 | 0.85 |
| 14 | ♀* | 49 | 1.64 | 8 | 4.84 | 9.67 | 95 | 3.92 | 1.05 | 0.81 |
| 15 | ♀ | 44 | 1.60 | 6 | 4.66 | 7.98 | 79 | 3.70 | 1.46 | 0.24 |

BMR, basal metabolic rate; Δ, change; LBM, lean body mass.

* More than twenty cigarettes daily.

† Viral respiratory infection, without fever, during second week of overfeeding.

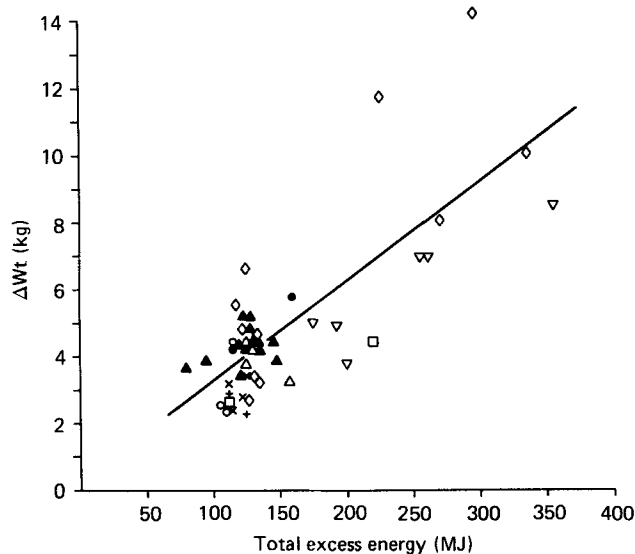


Fig. 1. Plot of observed change (Δ) in weight (kg) v. total excess energy (MJ):

$$y = 0.289 + 0.0297x \text{ (SE 1.6, } r \text{ 0.77).}$$

(●), (▲), Present values (two males, thirteen females); (○), from Passmore *et al.* (1955*a, b*) and Wiley & Newburgh (1931) (three males); (□), (△), from Miller & Mumford (1967*a, b*) (two males, three females); (◇), from Goldman *et al.* (1975) (thirteen males); (▽), from Norgan & Durnin (1980) (six males); (×), (+), from Webb & Annis (1983) (four males, two females).

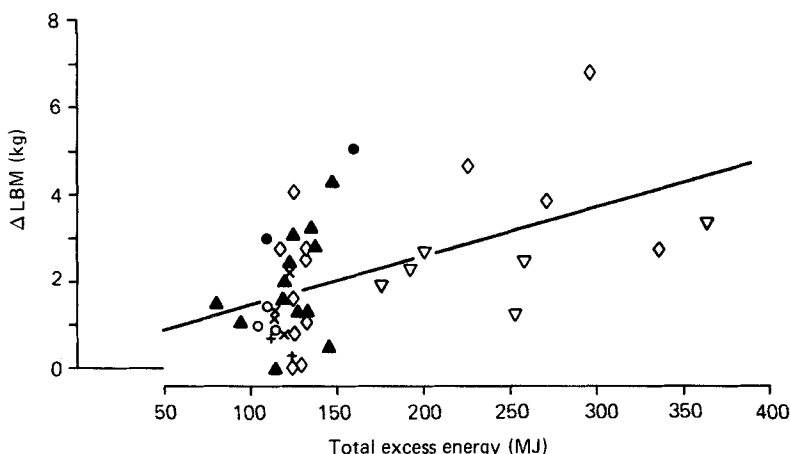


Fig. 2. Plot of observed change (Δ) in lean body mass (LBM; kg) v. total excess energy (MJ):

$$y = 0.332 + 0.0114x \text{ (SE 1.3, } r \text{ 0.49).}$$

(●), (▲) Present values (two males, thirteen females); (○), from Passmore *et al.* (1955*a, b*) and Wiley & Newburgh (1931) (three males); (◇), from Goldman *et al.* (1975) (thirteen males); (▽), from Norgan & Durnin (1980) (six males); (×), (+), from Webb & Annis (1983) (four males, two females). Goldman *et al.* (1975), Norgan & Durnin (1980), and Webb & Annis (1983) used densitometry; Passmore *et al.* (1955*a, b*) and Wiley & Newburgh (1931) used nitrogen balance, and the present study used ^{40}K counting to estimate changes in LBM. The regression of Δ weight on total excess energy for these forty-three subjects is:

$$y = 0.275 + 0.0305x \text{ (} r \text{ 0.79).}$$

Fig. 2 shows the changes in LBM induced by overfeeding. While the correlation coefficient was significant ($P < 0.01$), it was not as high as that for the weight gain values shown in Fig. 1; nevertheless the y -axis intercept was also close to zero. Three subjects failed to experience a gain in LBM, and in another the gain in LBM exceeded the gain in weight by a small margin. The reason for these unusual values and for the somewhat low correlation coefficient lies in the relative lack of precision of body composition measurement techniques compared with the scales used for measuring body-weight. For the forty subjects who had body-composition assays the average increment in LBM was 2.1 kg, which represents an increase of only about 4%, while the technical error of the assay procedure is at least 2%.

Using individual values for all forty-eight subjects, the mean energy cost of weight gain was 33.7 (SD 8.96) kJ/g (8.05 (SD 2.14) kcal/g), and the mean proportion of weight gain due to LBM was 0.436 (SD 0.290) (n 43). The coefficients of variation were 27 and 66% respectively. In an effort to discern the reason(s) for this variation in response, the subjects were grouped in various categories for further analysis. The categories selected were sex, initial body-weight, initial body fat content, type of excess food consumed, duration of overfeeding, daily excess energy consumption and smoking behaviour (Table 2). One-way analysis of variance within each category showed that there was no significant difference among the subgroups in any category. Of some interest is the finding that the four female subjects studied by us and who smoked more than twenty cigarettes daily throughout the study period had about the same response to overfeeding as the nine female non-smokers.

DISCUSSION

Although some of the findings reported here were not unexpected, three features stand out as being of interest. The first is that all the subjects, without exception, gained weight when overfed in a controlled environment, and the second is that there was a significant positive

Table 2. Examination of possible influence of subject characteristics
(Mean values and standard deviations)

| | Δ Wt/total excess energy (g/MJ) | | | Δ LBM/ Δ wt (g/g) | | |
|-----------------------------|--|------|----------|---------------------------------|-------|----------|
| | Mean | SD | <i>n</i> | Mean | SD | <i>n</i> |
| Sex | | | | | | |
| ♀ | 33.1 | 7.46 | 18 | 0.404 | 0.327 | 15 |
| ♂ | 31.1 | 9.56 | 30 | 0.453 | 0.273 | 28 |
| | $F = 0.55, P = 0.53$ | | | $F = 0.27, P = 0.61$ | | |
| Body-wt (kg) | | | | | | |
| 46-59 | 32.4 | 7.76 | 16 | 0.339 | 0.137 | 14 |
| 60-79 | 31.5 | 9.41 | 25 | 0.466 | 0.324 | 22 |
| 80-118 | 31.8 | 10.1 | 7 | 0.599 | 0.356 | 7 |
| | $F = 0.05, P = 0.96$ | | | $F = 1.99, P = 0.15$ | | |
| Body fat (kg) | | | | | | |
| < 10 | 34.5 | 11.2 | 15 | 0.400 | 0.219 | 15 |
| 10-20 | 33.0 | 7.32 | 17 | 0.429 | 0.325 | 17 |
| 21-44 | 29.0 | 7.08 | 8 | 0.537 | 0.373 | 8 |
| | $F = 1.02, P = 0.37$ | | | $F = 0.56, P = 0.58$ | | |
| Type of excess food | | | | | | |
| Carbohydrate | 32.3 | 9.97 | 12 | 0.425 | 0.351 | 12 |
| Fat | 37.0 | 13.2 | 5 | 0.477 | 0.191 | 5 |
| Mixed | 30.8 | 7.51 | 31 | 0.433 | 0.284 | 26 |
| | $F = 1.07, P = 0.35$ | | | $F = 0.06, P = 0.94$ | | |
| Duration of overfeeding (d) | | | | | | |
| < 31 | 32.0 | 8.34 | 38 | 0.444 | 0.323 | 33 |
| 31-83 | 31.4 | 10.9 | 10 | 0.409 | 0.140 | 10 |
| | $F = 0.03, P = 0.85$ | | | $F = 0.11, P = 0.74$ | | |
| Excess energy (MJ/d) | | | | | | |
| 3.6-5 | 31.6 | 10.7 | 15 | 0.385 | 0.169 | 14 |
| 5.1-6.9 | 32.7 | 6.51 | 17 | 0.477 | 0.302 | 14 |
| 7.0-9.2 | 31.2 | 9.53 | 16 | 0.445 | 0.369 | 15 |
| | $F = 0.13, P = 0.88$ | | | $F = 0.35, P = 0.71$ | | |
| Smoking behaviour* | | | | | | |
| Non-smokers | 35.2 | 6.95 | 9 | 0.465 | 0.396 | 9 |
| Smokers | 36.5 | 3.70 | 4 | 0.386 | 0.166 | 4 |
| | $F = 0.11, P = 0.74$ | | | $F = 0.14, P = 0.71$ | | |

Δ , Change; LBM, lean body mass.

* Females only.

correlation between the total excess energy consumed and the magnitude of the weight gain. The third is that the increment in LBM was also correlated with the total excess energy consumed.

While the general trends shown in Figs. 1 and 2 are evident, there was a fair amount of individual variability in the responses. The compilations shown in Table 2 show that this variability cannot be accounted for on the basis of sex, initial body-weight or body fat content, type of food consumed, duration of overfeeding, or the amount of food eaten each day; smoking behaviour did not influence the response of our female subjects. Reports by other investigators do not include information on smoking behaviour.

The lack of an influence of type of food consumed is in keeping with the observations of Fletcher *et al.* (1961) who found that type of food had no effect on the magnitude of

weight loss in obese subjects on weight-reduction diets. Claims of a sex difference in ease of weight gain or that overweight individuals gain weight more readily than thin individuals cannot be substantiated by the available findings (however, none of the forty-eight subjects was massively obese). Indeed, one of our subjects (weight 49 kg) stated that she had 'tried without success' to gain weight for several years; a similar claim was made by two of the males (weight 58 and 59 kg) studied by Passmore *et al.* (1955*a, b*). These three subjects actually gained as readily as the others. Gulick (1922) claimed to be a 'difficult fattening type,' yet an analysis of his findings showed that he always gained weight when he ate more food (Forbes, 1984). One of our female subjects made the interesting comment: 'I had always thought that I gained weight very easily, but I actually had to work very hard to put on those extra 10 pounds (4.46 kg) in 3 weeks.'

Possible reasons for the variability in the response to overfeeding include technical errors in food preparation and in designating the energy values of the various foods, errors in measurement of body composition and body-weight, variations in physical activity, errors in assessing maintenance energy requirements and hence in calculating excess energy intake. Obviously maintenance requirements should increase as body-weight is gained during overfeeding; however, only ten of the forty-eight subjects shown in Fig. 1 gained more than 10% of their initial weight, so this could not have been a large source of error for the majority of the subjects.

The possible influence of individual variations in thermogenesis cannot be evaluated from the available findings. Miller & Mumford (1967*a, b*) found an increase in 24 h metabolic rate in their subjects (but no consistent change in BMR), yet only one of their five subjects shown in Fig. 1 deviated significantly in weight response from the general trend shown. Twelve of our fifteen subjects exhibited a rise in BMR in response to overfeeding, and there was a correlation between the rise in BMR and the increase in body-weight; however, we do not have estimates of 24 h energy expenditure. In their carefully done studies of overfeeding, Dallosso & James (1984*a, b*) found 'only a small thermogenic component in excess of that anticipated for the energy costs of fat deposition'; and in their detailed review, Hervey & Tobin (1982) state: 'To the best of our knowledge, no measurements of energy expenditure have been reported, from humans or animals, that show clear evidence of *luxuskonsumption*', an opinion in accord with those of Wiley & Newburgh (1931) and Garrow (1978). The possibility exists that such effects could occur at times longer than the 83 d maximum period of overfeeding shown in Fig. 1, but to our knowledge there are no findings available for longer periods of controlled overfeeding. Since maintenance energy requirement is a function of body-weight (Jéquier & Schultz, 1983), one could anticipate that overfed subjects kept on a given level of energy intake would gain less weight as body-weight continued to increase, to reach finally a value for which the diet represented a maintenance requirement. For long periods of overfeeding, therefore, the rate of weight gain would be expected to decline in curvilinear fashion as times goes on.

Of interest is the finding that forty of the forty-three subjects who had body composition assays or complete N balance sustained an increase in LBM in response to overfeeding. Based on the observed ratio of the regression slopes for Figs. 1 and 2 ($0.0114/0.0297 = 0.384$), the average composition of the weight gain was 38% lean and 62% fat; based on means, these values are 44% lean and 56% fat. In earlier studies of adult men overfed for 6 months Keys *et al.* (1955) found by densitometry that 40% of their weight gain could be ascribed to LBM. Recently, Jen & Hansen (1983) reported that 34% of the weight gain in overfed monkeys was lean tissue. Spontaneous weight gain is also accompanied by an increase in LBM: observations (G. B. Forbes, unpublished results) on three obese adolescents who gained an average of 15 kg over a period of several months showed that LBM accounted for 40% of their weight gain; Sjöström (1980) restudied five

obese women who had gained an average of 10 kg over a period of several years, and found that LBM accounted for 31% of their weight gain. Unfortunately, there is no information as to which components of the LBM (viscera or muscle) participated in this increase; however, it is known that obese individuals have larger hearts, livers and kidneys than the non-obese (Naeye & Roode, 1970), as well as an increased urinary creatinine excretion (Tager & Kirsch, 1942) which is indicative of a larger muscle mass.

The average value of 38% of the weight gain as LBM found in the present study is higher than the percentage of excess weight contributed by LBM in adults with established obesity. In reviewing the literature, Forbes & Welle (1983) found an average value of 29% for the latter subjects (range 20–40%), and Webster *et al.* (1984) report values of 22–30% for obese females studied in their laboratory. This suggests that in the initial phase of overfeeding a somewhat higher proportion of the weight gain is non-fat material than that after prolonged overfeeding.

A question of some importance is how well the observed energy cost of the weight gain corresponds to the theoretical value derived from the composition of the gain. According to Spady *et al.* (1976) the cost of depositing 1 g fat is 50.2 kJ (12 kcal) and for 1 g protein it is 36.2 kJ (8.66 kcal); since the adult LBM contains 20.6% protein (Widdowson & Dickerson, 1964), the energy cost is 7.44 kJ (1.78 kcal)/g LBM. Based on the ratio of the regression slopes in Figs. 1 and 2 (0.384), the energy cost of the weight gain in this series of subjects was:

$$7.44 \times 0.384 \text{ g LBM} + 50.2 \times (1 - 0.384) \text{ g fat} = 33.8 \text{ kJ (8.08 kcal)/g weight.}$$

This is very close to the value of 33.7 kJ (8.05 kcal)/g derived from the regression line in Fig. 1. Comparable results are also found when one uses mean energy cost of weight gain (33.7 kJ (8.05 kcal)/g) and mean change in LBM: change in weight (0.436). Substituting this latter value in the equation gives

$$7.44 \times 0.436 + 50.2 \times (1 - 0.436) = 31.6 \text{ kJ (7.55 kcal)/g}$$

which is only 7% removed from the observed value. Viewed in another way, this excellent correspondence suggests that the energy-cost values for protein and fat chosen by Spady *et al.* (1976) are very close to the mark.

In summary, the results of our analysis show that substantial overfeeding under controlled conditions always induces weight gain, whether the subjects are initially thin or slightly obese, whether they be male or female; that the weight gain is proportional to the total amount of excess energy consumed; and that a substantial portion of the gain represents lean tissue. A problem worthy of further study is the elucidation of the mechanism(s) which facilitate the increase in LBM induced by excessive food intake.

REFERENCES

- Cuthbertson, D. P., McCutcheon, A. & Munro, H. N. (1937). *Biochemical Journal* **31**, 681–705.
 Dalosso, H. & James, W. P. T. (1984a). *British Journal of Nutrition* **52**, 49–64.
 Dalosso, H. & James, W. P. T. (1984b). *British Journal of Nutrition* **52**, 65–72.
 Fletcher, R. E., McCririch, M. Y. & Crooke, A. C. (1961). *British Journal of Nutrition* **15**, 53–58.
 Forbes, G. B. (1984). *American Journal of Clinical Nutrition* **39**, 349–350.
 Forbes, G. B., Schultz, F., Cafarelli, C. & Amirhakimi, G. H. (1968). *Health Physics* **15**, 435–442.
 Forbes, G. B. & Welle, S. L. (1983). *International Journal of Obesity* **7**, 99–107.
 Garrow, J. S. (1978). *Energy Balance and Obesity in Man*, 2nd ed. Amsterdam: Elsevier/North Holland
 Goldman, R. F., Haisman, M. F., Bynum, G., Horton, E. S. & Sims, E. A. H. (1975). In *Obesity in Perspective*, DHEW Publication no. (NIH)75-708, pp. 165–186 [G. A. Bray, editor]. Washington: DHEW.
 Gulick, A. (1922). *American Journal of Physiology* **60**, 371–395.
 Hervey, G. R. & Tobin, G. (1982). *Proceedings of the Nutrition Society* **41**, 137–153.
 Jen, K.-L. C. & Hansen, B. C. (1983). *Abstracts of the 4th International Congress on Obesity*, p. 14A.

Overfeeding in man

9

- Jéquier, E. & Schultz, Y. (1983). *American Journal of Clinical Nutrition* **38**, 989–998.
- Keys, A., Anderson, J. T. & Brozek, J. (1955). *Metabolism* **4**, 427–432.
- Miller, D. S. & Mumford, P. (1967*a*). *American Journal of Clinical Nutrition* **20**, 1212–1222.
- Miller, D. S. & Mumford, P. (1967*b*). *American Journal of Clinical Nutrition* **20**, 1223–1229.
- Naeye, R. L. & Roode, P. (1970). *American Journal of Clinical Pathology* **54**, 251–253.
- Norgan, N. G. & Durnin, J. V. G. A. (1980). *American Journal of Clinical Nutrition* **33**, 978–988.
- Olefsky, J., Crapo, P. A., Ginsburg, H. & Reaven, G. M. (1975). *Metabolism* **24**, 495–503.
- Passmore, R., Meiklejohn, A. P., Dewar, A. D. & Thow, R. K. (1955*a*). *British Journal of Nutrition* **9**, 20–27.
- Passmore, R., Meiklejohn, A. P., Dewar, A. D. & Thow, R. K. (1955*b*). *British Journal of Nutrition* **9**, 27–37.
- Sjöström, L. (1980). In *Obesity*, pp. 86–100 [A. J. Stunkard, editor]. Philadelphia: W. B. Saunders Co.
- Spady, D. W., Payne, P. R., Picou, D. & Waterlow, J. C. (1976). *American Journal of Clinical Nutrition* **29**, 1073–1088.
- Tager, B. N. & Kirsch, H. W. (1942). *Journal of Clinical Endocrinology and Metabolism* **2**, 696–699.
- Webb, P. & Annis, J. F. (1983). *Human Nutrition: Clinical Nutrition* **37C**, 117–131.
- Webster, J. D., Hesp, R. & Garrow, J. S. (1984). *Human Nutrition: Clinical Nutrition* **38C**, 299–306.
- Widdowson, E. M. & Dickerson, J. W. T. (1964). In *Mineral Metabolism*, vol. 2, part A, Chapt. 17 [C. L. Comar and F. Bronner, editors]. New York and London: Academic Press.
- Wiley, F. H. & Newburgh, L. H. (1931). *Journal of Clinical Investigation* **10**, 733–744.
- World Health Organization (1973). *Energy and Protein Requirements. Technical Report Series no. 522*. Geneva: WHO.